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THE UNIVERSITY OF ALBERTA

THE EFFECT OF MATERNAL NUTRITION AND MECHANICAL STRESS ON THE
INCIDENCE OF OSTEOCHONDROSIS IN SWINE

by

BETH WALKER



A THESIS

SUBMITTED TO THE FACULTY OF GRADUATE STUDIES AND RESEARCH

IN PARTIAL FULFILMENT OF THE REQUIREMENTS FOR THE DEGREE

OF MASTER OF SCIENCE

IN

Animal Nutrition

Department of Animal Science

EDMONTON, ALBERTA

FALL 1987

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The undersigned certify that they have read, and recommend to the Faculty of Graduate Studies and Research, for acceptance, a thesis entitled THE EFFECTS OF MATERNAL NUTRITION AND MECHANICAL STRESS ON THE INCIDENCE OF OSTEOCHONDROSIS IN SWINE submitted by BETH WALKER in partial fulfilment of the requirements for the degree of MASTER OF SCIENCE in Animal Nutrition.

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Date *Aug 21, 1987*

ABSTRACT

Eighty crossbred pigs were selected from 20 third parity sows fed either an adequate or restricted (50% of adequate) level during lactation for three consecutive parities. Pigs were housed in groups of four and fed standard starter, grower and finisher diets (19, 16 and 14% crude protein, respectively) ad libitum until slaughter at 100 kg liveweight. Sow nutritional restriction did not significantly affect the incidence of osteochondrosis in the elbow joint of offspring. Pigs from restricted-fed sows had a higher incidence of osteochondrosis in the distal ulna physis (growth plate) than pigs from adequately fed sows. The uronic acid content (an indication of proteoglycan content) of articular cartilage from the medial humeral condyle was not significantly different between treatment groups.

Sixteen litters of eight crossbred pigs were randomly assigned to one of four groups composed of two pre-weaning handling treatments and one of three post-weaning mixing schedules. Pre-weaning treatments involved gentle and normal handling during the routine pre-weaning management practices. Post-weaning, pigs either remained as littermates or were mixed at weaning and 4 months of age or every month. All pigs were fed standard starter, grower and finisher diets ad libitum. Eight pigs from each treatment group were randomly selected for slaughter at 3, 4 and 5 months of age.

Pre-weaning handling and post-weaning mixing treatments did not significantly affect the incidence of osteochondrosis at any stage of the experiment. Scanning electron microscopy showed detrimental effects of mixing on the surface morphology of articular cartilage (osteoarthrosis) in the knee and elbow joints by six months of age.

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I. INTRODUCTION

The biochemical composition (Mc Devitt and Muir, 1976) and ultrastructural organisation (Bhatnager et al., 1981; Farnum and Wilsman, 1986) of components in hyaline articular cartilage has been extensively reviewed (Nakano et al., 1981a; Nakano et al., 1987a; Poole, 1986) and changes in normal cartilage with age and the onset of osteochondrosis have been described (Nakano et al., 1979a). This chapter will therefore be confined to a description of the data specifically related to the experiments described herein. Studies of the articular cartilage of pigs have revealed that growing- finishing pigs are highly susceptible to osteochondrosis with advancing age (Grondalen, 1974a, b). The clinical symptoms of the disease are evident by about six months of age when severe degeneration of joint cartilage is an important cause of leg weakness (Grondalen, 1974c; Reiland, 1978a). Since lameness is a major reason for culling breeding stock on commercial farms (Friendship et al., 1986) it is vital that we gain an understanding of the etiology of osteochondrosis.

Osteochondrosis is a non-infectious degenerative joint disease affecting growing animals, including pigs, humans, dogs, cattle, poultry and rats (Reiland, 1978a; Siffert, 1981). The disease is characterised by disruption of normal endochondral ossification; the mechanism whereby cartilage is transformed into bone. Bone growth is accomplished by cartilage cell proliferation and matrix production, metabolic elaborations rendering the matrix calcifiable, followed by

calcification. Replacement of the degenerating cartilage cells occurs by osteoid material originating from invading metaphyseal vascular elements and finally, new bone is formed on calcified growth cartilage remnants (Siffert, 1981).

Composition of normal cartilage

Articular epiphyseal and physeal (growth-plate) cartilage is composed of chondrocytes embedded in an extracellular matrix of water (60-80%), collagen and proteoglycans. The specific arrangement of type II collagen fibres within the different zones of cartilage provides tensile strength while proteoglycan aggregates confer compressive strength by virtue of their water binding capacity (Muir, 1977; Mow et al., 1984). In addition, intermolecular interactions between cartilage macromolecules are hypothesised as being central to the structural and functional integrity of cartilage (Muir, 1977). Proteoglycans function in the binding of large amounts of water, providing the cartilage with its elastic weight bearing properties and are also important in the regulation of nutrient and waste product transport through the cartilage (Poole, 1986). These properties are largely a consequence of the highly anionic nature of proteoglycan aggregates resulting from the high degree of sulfation in component glycosaminoglycans (chondroitin sulfate and keratin sulfate).

Proteoglycans in hyaline cartilage exist in both monomer and aggregate (predominant form) forms. Aggregation of monomers by non-covalent association with hyaluronic acid and link proteins is

considered to be significant in retaining proteoglycans in the matrix and degradation of aggregates seems to be important in the onset of calcification (Buckwater, 1983).

Tissue alterations in osteochondrosis

The specific changes associated with component proteoglycans and chondrocyte biosynthesis are central to the understanding of osteochondrosis. The earliest macroscopic evidence of osteochondrosis in pigs occurs at around 10 weeks of age and is seen as the retention of epiphyseal cartilage in subchondral bone due to a failure in endochondral ossification (Nakano et al., 1979a,b). Recent ultrastructural and histological observations have shown morphological evidence of lesions characteristic of osteochondrosis in the cartilage of piglets at birth (Hill et al., 1985). It is unclear as yet whether the primary changes in osteochondrosis originate in the cartilage or in the bone since it is not known what causes either the failure of endochondral ossification or the accompanying failure of vascular invasion. More recent evidence suggests a cartilaginous origin (Nakano et al., 1987b), and it has been suggested that the disorder may result from abnormal growth and maturation of chondrocytes (Olsson, 1978).

The early lesions described as a thickening in the zone of hypertrophying chondrocytes are thought to progress with age into degenerative joint disease (Hill et al., 1985). As bone growth advances the lesion area becomes enlarged and may ultimately result in separation of cartilage from subchondral bone (osteochondrosis dissecans). Lesion areas are associated with reduced proteoglycan content and aggregating ability and hence increased extractability of

proteoglycans from the cartilage (Nakano et al., 1985). A reduction in proteoglycan content has been demonstrated by reduced uronic acid content (a component sugar of chondroitin sulfate) and histologically as a reduction in the intensity of proteoglycan staining (safranin-O) (Nakano et al., 1979a, b). Surface erosion (osteoarthrosis) and proteoglycan loss (in the superficial zone) is also frequently observed secondary to osteochondrosis. The surface morphology of normal and osteochondrotic articular cartilage in pigs has recieved little attention. Scanning electron microscopy has been used in a number of species to examine normal and degenerative surface morphology of joint cartilage (Redler, 1974; Walton, 1977) and may provide a useful tool for the study of articular cartilage changes in the knee and elbow joints of pigs. There is little information on the surface morphology of normal and osteochondrotic porcine cartilage.

The inability of proteoglycans to form aggregates in osteochondrotic cartilage may be due to a number of factors, such as a defect in the components synthesised eg. the hyaluronic acid binding region of core proteins, or enzymatic degradation of core protein eg. by neutral metalloproteases (Ali and Evans, 1973). The reduction in proteoglycan content is accompanied by increased water loss and a reduction in the compressive strength of the cartilage which may pre-dispose it to further damage (Mow et al., 1984).

In pigs at birth lesions occur randomly across sites in the limb joints and other sites of the body (Hill et al., 1985) but with increasing age there is an increase in the incidence and severity of lesions at specific sites in the knee and elbow joints. The sites most severely affected with degenerative joint disease are the distal ulna

physis and the articular cartilage of the distal femur and humerus, in particular the medial condyles (Grondalen 1974a, b; Nakano et al., 1981b). Hence, although the more recent information suggests a congenital condition (Hill et al., 1985) there appear to be factors associated with exacerbating the development of lesions in the knee and elbow joints.

Etiology

Nutrition. The observations of a low incidence of osteochondrosis in wild pigs (Fell et al., 1967) and an absence of the disease in Yucatan (slow-growing) pigs (Farnum and Willsman, 1986) have prompted suggestions that intensive confinement-rearing conditions are important factors in the high incidence of osteochondrosis in commercial breeds of pigs. Nutrition of the growing-finishing pig has received much attention particularly with respect to the rapid weight gains achieved under ad libitum conditions. It has been suggested that the extremely rapid rates of liveweight gain of commercial pigs creates too much weight stress for immature joint cartilage to bear and that a reduction in feed intake may reduce the incidence of osteochondrosis (Reiland, 1978b). In general, feed intake restrictions and subsequent reductions in growth rate have not had a preventative effect on the development of osteochondrosis (Grondalen, 1974c, d; Hanssen and Grondalen, 1979; Nakano et al., 1979b, 1984a), though genetically selected slow-growing lines of pigs have been reported to have a slightly lower incidence than fast-growing lines (Grondalen and Vangen, 1974).

The deficiency symptoms of a number of minerals and vitamins

involve disturbances in bone and cartilage or disturbance of locomotory ability and have therefore prompted investigation. Variation in dietary calcium and phosphorus levels (important in bone mineralisation) (Brennan and Aherne, 1986; Grondalen, 1974d) and manipulation of the amount of a number of other minerals (manganese, magnesium, copper and zinc) (Walker et al., 1966) in the diets of growing pigs have had no influence on the incidence of osteochondrosis. In addition, levels of calcium and phosphorus in subchondral bone adjacent to lesion areas appeared to be normal (Nakano et al., 1981b).

Vitamin C (ascorbic acid) is important in the enzymatic formation of crosslinks and hydrogen bonding in collagen molecules (by hydroxylation of proline and lysine), which provide stability and rigidity in the cartilage. Serum and urinary levels of ascorbic acid were normal in pigs with osteochondrotic lesions (Walker et al., 1966) and supplementary levels of vitamin C had no effect on the development of osteochondrosis (Nakano et al., 1983). The collagen content of osteochondrotic cartilage and the content of the non-reducible intermolecular crosslink pyridinoline have been shown to approach those in normal cartilage (Nakano et al., 1984b). Variation in dietary levels of vitamins A, D (Reiland, 1978c) and E (Grondalen, 1974c) have not had any preventative effect on the incidence of the disease.

Manipulation of nutrient intake in growing-finishing pigs has been unsuccessful as a preventative method in the development of osteochondrosis (Nakano et al., 1979b). The appearance of lesions in piglets at birth suggests that if nutrition is to be implicated then maternal nutrition should be examined (Hill et al., 1985). It has been suggested that maternal nutrition may influence the process of

endochondral ossification or chondrocyte maturation in the developing fetus. It is known that the latter part of pregnancy (and fetal development) and the early weeks of piglet life are important periods in the development of articular cartilage in pigs, since the most marked changes in biochemical composition and cell biosynthesis occur at this time (Nakano et al., 1979a, b; Simunek and Muir, 1972). No studies have been carried out to examine the involvement of sow maternal nutritional factors in the onset of osteochondrosis in their offspring.

Environment. Environmental factors such as lack of exercise and floor type have also been implicated as important factors in the development of osteochondrosis in growing-finishing pigs (Elliot and Doige, 1973). Joint motion or exercise is considered important in the maintenance of cartilage integrity by promoting the diffusion of nutrients from the synovial fluid to the cartilage. In the mature animal, cartilage is avascular and is therefore dependant on the diffusion of nutrients from the synovial fluid. Since pigs spend approximately 80% of their time inactive (particularly under individually penned conditions) it has been suggested that lack of exercise may be responsible for the degeneration of joint cartilage (Elliot and Doige, 1973).

Immobilisation of the joints in a plaster cast has been used to demonstrate cartilage degeneration with cessation of normal movement (Caterson and Lowther, 1978; Palmoski et al., 1979, 1980). Varying levels of exercise have been employed to study the response of cartilage to movement and joint loading in a number of species. Long term treadmill exercise of pigs was shown to improve locomotary ability

but had no effect on the incidence of osteochondrotic lesions (Perrin and Bowland, 1977). More extreme exercise regimes in dogs have been shown to cause changes in matrix synthesis and degenerative changes in articular cartilage (Vasan, 1983).

A number of studies indicate that articular cartilage chondrocyte biosynthesis is responsive to different loading or mechanical pressure conditions without degeneration occurring between as yet unquantified physiological threshold limits (Dekel and Weissman, 1978; Palmoski and Brandt, 1984; Lipiello et al., 1985). Outside these limits degeneration of cartilage occurs.

It has frequently been suggested that the cartilage of rapidly growing pigs is at the upper limit of its weight bearing capacity and that mechanical stresses on the joints such as the transportation or mixing of pigs would be sufficient to cause traumatic insult and subsequent degeneration of cartilage (Grondalen, 1977; Nakano et al., 1981a). In support of this hypothesis are the prevalence of lesions on the weight bearing regions of the medial condyles of the femur and humerus and visual signs of wear on the proximal radius (Nakano et al., 1984a). In addition most recent evidence from work with human osteochondroses and particularly osteoarthrosis suggests a traumatic origin for these degenerative joint diseases (Siffert, 1981).

Further support for the involvement of mechanical trauma in the etiology of osteochondrosis comes from experiments involving overloading of the knee and elbow joints of pigs. Immobilisation of one foreleg, used to put excessive weight on the contralateral limb, and loading pigs with lead saddles (at 15% of bodyweight) have both been shown to accelerate the development of osteochondrotic lesions in the

articular cartilage and in the distal ulna physis of pigs (Walker et al., 1966; Grondalen and Grondalen, 1974; Brennan et al., 1985).

However, in these experiments osteochondrotic lesions were also observed in the control (non-mechanically stressed) animals suggesting a more complex etiology for the onset of the disease.

Other factors such as conformation, carcass characteristics, joint instability, breed and inheritability have been proposed to play a role in the etiology of osteochondrosis, but in general osteochondrosis occurs in any breed or sex (Goedegeburre et al., 1980) and the inheritability of leg soundness score has been reported as low to moderate (Webb, 1983).

The objectives of the studies described herein were to examine the effects of maternal nutrition and the mechanical stress of mixing on the incidence and severity of osteochondrosis and the surface morphology of articular cartilage.

A. BIBLIOGRAPHY

- Ali, S.Y., and Evans, L. 1973. Enzymatic degradation of cartilage in osteoarthritis. *Fed. Proc.* 32:1494-1498.
- Bhatnager, R., Christian, R.G., Nakano, T., Aherne, F.X., and Thompson, J.R. 1981. Age related changes in osteochondrosis in swine articular and epiphyseal cartilage: light and electron microscopy. *Can. J. Comp. Med.* 45: 188-195.
- Brennan, J.J., Aherne, F.X., Thompson, J.R., and Nakano, T. 1985. Effect of increased weight stress on joint integrity and Cathepsin B-like activity of articular cartilage in growing swine. *Proc. Swine. in Biomed. Res. Conf.*
- Brennan, J.J., and Aherne, F.X. 1986. Effect of dietary calcium and phosphorus levels on performance, bone bending moment and the severity of osteochondrosis and lameness in boars and gilts slaughtered at 100 or 130 kg body weight. *Can. J. Anim. Sci.* 66: 777-790.
- Buckwalter, J.A. 1983. Proteoglycans and calcifying cartilage. *Clin. Orthop.* 179: 207-232.
- Caterson, B., and Lowther, D.A. 1978. Changes in the metabolism of the proteoglycans from sheep articular cartilage in response to mechanical stress. *Biochem. Biophys. Acta.* 540: 412-422.
- Dekel, S., and Weissman, S.L. 1978. Joint changes after overuse and peak overloading of rabbit knees in vivo. *Acta. Orthop. Scand.* 49: 519-528.
- Elliot, J.I., and Doige, C.E. 1973. Effects of type of confinement on performance and on the occurrence of locomotory disturbances in market pigs. *Can. J. Anim. Sci.* 53: 211-217.

- Farnum, C.E., and Wilsman, N.J. 1986. Ultrastructural histochemical evaluation of growth plate cartilage matrix from healthy and osteochondrotic swine. *Am. J. Vet. Res.* 47: 1105-1115.
- Fell, B.F., Jones, A.S., and Boyne, R. 1967. Lesions of the distal ulna epiphysis in Wild East African pigs. *Vet. Rec.* 81:341.
- Friendship, R.M., Wilson, M.R., Almond, G.W., McMillan, J., Hacker, R.R., Pieper, R., and Swaminathan, S.S. 1986. Sow wastage: Reasons for and effect on productivity. *Can. J. Vet. Res.* 50: 205-208.
- Goedegeburre, S.A., Hani, H.J., Van Der Valk, P.C., and Van Der Wal, P.G. 1980. Osteochondrosis in six breeds of slaughter pigs. I. A morphological investigation of the status of osteochondrosis in relation to breed and level of feeding. *Vet. Q.* 2: 28-41.
- Grondalen, T. 1974a. Osteochondrosis and arthrosis in pigs. I. Incidence in animals up to 120 kg liveweight. *Acta. Vet. Scand.* 15: 1-25.
- Grondalen, T. 1974b. Osteochondrosis and arthrosis in pigs II. Incidence in breeding animals. *Acta. Vet. Scand.* 15: 226-242.
- Grondalen, T. 1974c. Leg weakness in pigs. I. Incidence and relationship to skeletal lesions, feed level, protein and mineral supply, exercise and exterior conformation. *Acta. Vet. Scand.* 15: 555-573.
- Grondalen, T. 1974d. Osteochondrosis and arthrosis in pigs. VI. Relationship to feed level and calcium, phosphorus and protein levels in the ration. *Acta. Vet. Scand.* 15: 147-169.
- Grondalen, T., and Grondalen, G. 1974. Osteochondrosis and arthrosis in pigs. IV. Effects of overloading on the distal epiphyseal plate of the ulna. *Acta. Vet. Scand.* 15: 153-160.

- Grondalen, T., and Vangen, O. 1974. Osteochondrosis and arthrosis in pigs. V. A comparison of the incidence in three different lines of Norwegian landrace breed. *Acta. Vet. Scand.* 15: 61-79.
- Grondalen, T. 1977. Viewpoints on the porcine leg weakness syndrome. *Proceedings of the 3rd Int. Conf. on Production Diseases in Farm Animals. Wageningen, The Netherlands. 1976.* pp. 214-217.
- Hanssen, J.T., and Grondalen, T. 1979. The effect of different Dietary factors on growth and development in young boars and gilts. *Z. Tierphysiol. Tierernahrg u Futtermittelkde.* 42: 65-83.
- Hill, M.A., Ruth, G.R., Hilley, H.D., Torrison, J.L., Bagent, J.K., and Leman, A.D. 1985. Dyschondroplasias of growth cartilages (osteochondrosis) in crossbred commercial pigs at one and fifteen days of age: Radiological, angiomicrographical and histological findings. *Vet. Rec.* 116: 40-47.
- Lipiello, L., Kaye, C., Neumata, T., Mankin, H.J. 1985. In vitro metabolic response of articular cartilage segments to low levels of hydrostatic pressure. *Conn. Tissue. Res.* 13: 99-107.
- McDevitt, C.A., and Muir, H. 1976. Biochemical changes in the cartilage of the knee in experimental and natural osteoarthritis in the dog. *J. Bone. Joint. Surg.* 58B: 94-101.
- Mow, C., Holmes, M.H., and Lai, M. 1984. Fluid transport and mechanical properties of articular cartilage: A review. *J. Biomechanics.* 17: 377-394.
- Muir, H. 1977. Molecular approach to the understanding of osteoarthritis. *Ann. Rheum. Dis.* 36: 199-208.
-

- Nakano, T., Aherne, F.X., and Thompson, J.R. 1979a. Changes in swine knee articular cartilage during growth. *Can. J. Anim. Sci.* 59: 167-169.
- Nakano, T., Aherne, F.X., and Thompson, J.R. 1979b. Effects of feed restriction, sex and diethylstilbestrol on the occurrence of joint lesions with some histological and biochemical studies of the articular cartilage of growing-finishing swine. *Can. J. Anim. Sci.* 59: 491-502.
- Nakano, T., Aherne, F.X., and Thompson, J.R. 1981a. Leg weakness and osteochondrosis in pigs. *Pig. News. Info.* 2: 29-34.
- Nakano, T., Aherne, F.X., and Thompson, J.R. 1981b. Mineralisation of normal and osteochondrotic bone in swine. *Can. J. Anim. Sci.* 61: 343-348.
- Nakano, T., Aherne, F.X., and Thompson, J.R. 1983. Effect of dietary supplementation of Vitamin C on pig performance and the incidence of osteochondrosis in elbow and stifle joints in young growing swine. *Can. J. Anim. Sci.* 63: 421-428.
- Nakano, T., Aherne, F.X., Brennan, J.J., and Thompson, J.R. 1984a. Effect of growth rate on the incidence of osteochondrosis in growing swine. *Can. J. Anim. Sci.* 64: 134-146.
- Nakano, T., Thompson, J.R., Aherne, F.X., and Christian, R.G. 1984b. Non-reducible collagen crosslink in normal and degenerative porcine joint cartilage. *Biomed. Res.* 5: 371-374.
- Nakano, T., Brennan, J.J., and Aherne, F.X. 1987a. Leg weakness and osteochondrosis in swine: a review. *Can. J. Anim. Sci.* (in press).
- Nakano, T., Aherne, F.X., Brennan, J.J., and Christian, R.G. 1987b. Observations on osteochondrosis in stifle joints from five to 20

- week old swine. *Can. J. Anim. Sci.* (in press).
- Nakano, T., Thompson, J.R., and Aherne, F.X. 1985. Cartilage proteoglycans from normal and osteochondrotic porcine joints. *Can. J. Comp. Med.* 49: 219-226.
- Olsson, S.E. 1978. Osteochondrosis in domestic animals. *Acta. Radiol. Suppl.* 358: 9-14.
- Palmoski, M.J., Perricone, E., and Brandt, K.D. 1979. Development and reversal of a proteoglycan aggregation-defect in normal canine knee cartilage after immobilisation. *Arth. Rheum.* 22: 508-517.
- Palmoski, M.J., Colyer, R.A., and Brandt, K.D. 1980. Joint motion in the absence of normal loading does not maintain normal articular cartilage. *Arth. Rheum.* 23: 325-340.
- Palmoski, M.J., and Brandt, K.D. 1984. Effects of static and cyclic compressive loading on articular cartilage plugs in vitro. *Arth. Rheum.* 27: 675-681.
- Perrin, W.R., and Bowland, J.P. 1977. Effects of enforced exercise on the incidence of leg weakness in growing boars. *Can. J. Anim. Sci.* 57: 245-253.
- Poole, A.R. 1986. Proteoglycans in health and disease: structure and functions. *Biochem. J.* 236:1-14.
- Redler, I. 1974. A scanning electron microscope study of human normal and osteoarthritic articular cartilage. *Clin. Orthop.* 103:262-268.
- Reiland, S. 1978a. Pathology of so called leg weakness in the pig. *Acta. Radiol. (Suppl)*, 358: 23-44.
- Reiland, S. 1978b. The effect of decreased growth rate on frequency and severity of osteochondrosis in pigs. *Acta. Radiol. (Suppl)*, 358: 107-122.

- Reiland, S. 1978c. Effects of Vitamin D and A, calcium, phosphorus and protein on frequency of osteochondrosis in pigs. An experimental investigation. *Acta. Radiol. (Suppl)*, 358: 107-122.
- Siffert, R.S. 1981. Classification of the osteochondroses. *Clin. Orthop. Rel. Res.* 158: 10-18.
- Simunek, Z., and Muir, H. 1972. Changes in the protein-polysaccharides of pig articular cartilage during pre-natal life, development and old age. *Biochem. J.* 126: 515-523.
- Vasan, N. 1983. Effects of physical stress on the synthesis and degradation of cartilage matrix. *Conn. Tissue. Res.* 12: 49-58.
- Walker, T., Fell, B.F., Jones, A.S., Boyne, R., and Elliot, M. 1966. Observations on "leg weakness" in pigs. *Vet. Rec.* 79: 472-479.
- Walton, M. 1977. Studies of degenerative joint disease in the mouse knee joint; scanning electron microscopy. *J. Pathol.* 123 (4): 211.
- Webb, A.J., Russel, W.C., and Sales, D.I. 1983. Genetics of leg weakness in performance-tested boars. *Anim. Prod.* 36: 117-130.

II. THE EFFECT OF SOW NUTRITION DURING LACTATION ON THE INCIDENCE AND SEVERITY OF OSTEOCHONDROSIS IN THEIR OFFSPRING AT 100 KG LIVEWEIGHT

A. INTRODUCTION.

Osteochondrosis is a generalised non-infectious skeletal disorder associated with the joint cartilage (articular-epiphyseal cartilage complex) and physeal cartilage in growing pigs. Osteochondrosis also occurs in other species including dogs, horses and humans (Siffert, 1981; Reiland, 1978). The disorder is characterised by a disturbance of endochondral ossification resulting in cartilage retention in subchondral bone, (Siffert, 1981). The advanced stages are frequently associated with separation of cartilage from the underlying bone (osteochondrosis dissecans) and secondary osteoarthritis.

Osteochondrotic lesions have been observed in almost 100% of pigs by the age of six months and are considered to be a potential cause of clinical lameness in young, mature breeding stock, (Olsson and Reiland, 1978; Reiland, 1978; Grondalen, 1974a, b; Nakano et al., 1981a). Various aspects of commercial pig production have been implicated in the development of osteochondrosis in growing-finishing pigs. Confinement rearing problems such as lack of exercise, type of flooring (Perrin and Bowland, 1977; Perrin et al., 1978; Fredeen and Sather, 1978; Nakano et al., 1981b; Brennan and Aherne, 1987), growth rate (Nakano et al., 1979b, 1984; Fell et al., 1970), and nutrition (vitamins, minerals, energy and protein) (Walker et al., 1966; Grondalen, 1974c; Hanssen and Grondalen, 1979; Empel, 1980; Nakano et

al., 1979b, 1983a; Brennan et al., 1986) have been extensively studied as causative factors. Manipulation of nutrition and of growth rate generally appear to have no preventative effects on the development of osteochondrosis in growing-finishing pigs. However, a recent report indicating that lesions characteristic of osteochondrosis are visible histologically in pigs at birth (Hill et al., 1985), led these authors to suggest that maternal nutrition may be a factor in the development of osteochondrosis, though no studies have been conducted to examine this hypothesis in pigs. Two critical periods in the development and maturation of joint cartilage in pigs during which sow nutrition may be of importance are in the fetus during the second half of pregnancy and in the neonatal piglet during the first few weeks of life, when the most marked changes in cartilage composition occur (Brandt and Muir, 1969; Simunek and Muir, 1972a; Nakano et al., 1979a, 1982; 1983b; Bhatnagar et al., 1981).

This study was undertaken to elucidate the effects of nutritional restriction of sows on the occurrence of osteochondrotic lesions in their offspring when slaughtered at a weight of 100 kg.

B. MATERIALS AND METHODS

Eighty crossbred (Yorkshire X Landrace) pigs (39 barrows and 41 gilts) were selected from 20 third parity sows (crossbred) fed either an adequate (high feed intake) or restricted (low feed intake) level during lactation for three consecutive parities. During gestation all sows received 2.25 kg d^{-1} of a 16% crude protein (CP) diet which supplied 16.5 MJ kg^{-1} of digestible energy. At farrowing the sows

were fed a 16% CP diet to a scale which allowed feed intakes equal to 10 (week 1), 13 (week 2), and 14 (weeks 3 and 4) percent of metabolic body weight ($\text{kg}^{0.75}$) for sows fed an adequate level or 50% of these levels for restricted fed sows. The resulting average feed intakes over the four week lactation were 3 and 6 kg d^{-1} for low and high feed intake sows, respectively. The diet for restricted-fed sows was formulated to contain additional levels of vitamin-mineral premix so that only protein and energy intake of those sows was restricted (NAS-NRC, 1979). Piglets were not allowed access to creep feed during the lactation period.

At weaning pigs were randomly assigned to groups of four and were housed in 2.4 m X 1.5 m concrete floored pens with straw used as bedding. Pigs were fed standard starter, grower and finisher diets containing 19, 16 and 14 % CP, respectively. Water and feed were freely available throughout the experiment. Pig weights and feed intakes were recorded weekly from weaning to slaughter at 100 to 105 kg liveweight. On the day before slaughter locomotory ability was assessed by the author for each pig using a subjective score of 1 to 5 (1-normal, sound, 2-rotation of hind limbs, 3-slight limp (favouring of one limb), 4-distinct lameness, 5-severe lameness), (Nakano et al., 1983a).

Soundness of gait was recorded by walking pigs individually along a central isle of the barn several times. After slaughter the elbow joints were removed intact from both sides of the carcass. Joint cartilage and subchondral bone from the distal humerus including medial and lateral condyles, proximal radius and ulna, and distal ulna (physis) were visually appraised for soundness. Evidence of osteochondrosis was determined on a scale of 1 to 5 (1-normal and free

of lesions, 5=severe lesions), (Nakano et al., 1983a).

Transverse sections of 2 mm X 10 mm cartilage with subchondral bone were taken from the central weight bearing region of normal and osteochondrotic medial humeral condyles for histological examination and fixed in 10% phosphate-buffered formalin (pH 7.0). Tissues were then decalcified, fixed in paraffin (Drury et al., 1967) and 7 μ m thick sections were stained with safranin-O, fast green and iron haematoxylin (Rosenburg, 1971).

Cartilage samples were also taken for the determination of uronic acid, from the central weight bearing region of the medial humeral condyle of all normal condyles, and from the lesion area (if different from above) of osteochondrotic condyles. Samples were dried in acetone and then digested with papain (5 mg of twice crystalized enzyme per g of dry tissue) for 48 hours at 60° C, (McDevit and Muir, 1976). Digests were centrifuged and the supernatant diluted to 10 mL with water. Determination of uronic acid content by a modified carbazole reaction (Bitter and Muir, 1962) was performed on 100 μ L aliquots of supernatant.

Data were analysed by analysis of variance. Regression analysis (Steele and Torrie, 1980) was used to determine the relationship between uronic acid content and lesion score.

C. RESULTS

Sows in their third parity on the restricted feeding regime lost considerably more weight ($P<0.05$) over the four week lactation than sows on the high feed intake; 26 kg and 3 kg, respectively, and they also produced less milk ($P<0.05$). Individual amounts of milk produced

were estimated as 7.6 kg d^{-1} and 4.4 kg d^{-1} , respectively for the high and low fed sows, based on the litters growth rate (1 g gain=4 g milk). Milk composition for high and low fed sows was similar for protein and fat content, data not shown (Lithgoe, 1987). Pigs from the low feed intake sows were smaller at birth and weaning ($P < 0.01$), but had significantly larger rates of liveweight gain in the grower period (7 to 60 kg) than pigs from the sows fed a high feed intake level (Table II.1). The difference however was small (0.03 kg day^{-1}). Pigs from low feed intake sows were slightly heavier at slaughter as a result of being three days older.

Upon visual examination of elbow joints and distal ulna physes, 94% of the pigs studied showed signs of osteochondrotic lesions in one or more of the sites scored. Lesions occurred with most frequency and severity on the medial humeral condyle (44% of pigs affected) and in the physis of the distal ulna (61% incidence) in agreement with other studies (Grondalen, 1974a; Nakano et al., 1984). Lesions of osteochondrosis in the articular cartilage of the elbow were characterised by failure of endochondral ossification and cartilage retention in the subchondral bone. Severe lesions were often accompanied by cartilage separation from subchondral bone with frequent erosion of the cranio-medial edge of the proximal radius.

Histologically osteochondrotic lesions were manifested in a number of ways;

(i) focal areas of reduced proteoglycan staining with safranin-O were evidence of proteoglycan loss from lesion areas (Chapter III, Fig.2),

(ii) cell clusters arising around areas of cartilage fracture which may

be an attempt by chondrocytes to increase proteoglycan production in response to the tissue damage (Nakano et al., 1979a),

(iii) severe lesions were often associated with a focal area of chondrolysis, with apparently normal chondrocytes in the adjacent area,

(iv) cartilage cracks or separation of cartilage in the deep zone from the subchondral bone and failure of cartilage to ossify,

(v) blood cells in the bone were evidence of haemorrhage in very severe cases, and

(vi) presence of fibrous tissue as repair tissue was observed in both damaged cartilage and underlying bone. It is debatable as to how much repair occurs in cartilage and bone, and to what extent the scar tissue and hyaline cartilage progress and heal, (Ghadially et al., 1977). The gross and histological observations reported herein were similar to those reported previously (Grondalen, 1974a; Reiland, 1978; Nakano et al., 1979a, 1982; Bhatnager et al., 1981).

The incidence (% of pigs affected) and severity (lesion score) of osteochondral lesions in pigs from high and low feed intake sows were not significantly different. There were apparent ($P < 0.05$) differences between groups of pigs from high and low feed intake sows in average lesion score of the lateral humeral condyle, and proximal ulna (Table II.2), pigs from the high feed intake sows having the higher lesion scores. Pigs from the undernourished sows had a significantly higher average lesion score in the distal ulna physis ($P < 0.01$) than pigs from high feed intake sows. The lesion score at the distal ulna physis was also weakly correlated with individual maternal weight loss over the lactation period ($r = 0.16$, $P < 0.001$). Average lesion scores for sites in the elbow joint did not correlate ($P > 0.05$) with sow weight loss. There

were no sex differences in the locomotory ability or the incidence of osteochondrosis at any of the sites scored.

The uronic acid content of the medial humeral condylar cartilage for pigs from high and low feed intake sows are presented in Table II.3. The average uronic acid content of normal and osteochondrotic cartilage in this experiment was 5.19 % of dry weight. There were no significant differences in uronic acid content of the joint cartilage between sexes or between pigs from low-fed and high-fed sows. With increasing severity of lesion score there was an apparent reduction in uronic acid concentration of the cartilage, (Table II.4), but the correlation coefficient for cartilage uronic acid concentration and osteochondrotic lesion score was very low ($r=0.02$, $P>0.05$). Cartilage with lesion scores of 4 and 5 (severe osteochondrosis) showed a 16% reduction in uronic acid content compared to normal cartilage (lesion score=1) ($P<0.05$).

A large range of growth rates was observed in this experiment (0.65 to 0.95 kg.d^{-1}) and although growth rates differed ($P<0.05$) between pigs from high and low feed intake sows, average daily gain of offspring was not directly correlated with the extent of maternal weight loss. The observed differences in growth rate in this study were not related to locomotory ability or the incidence of osteochondrosis at any of the joint sites scored ($P>0.05$).

D. DISCUSSION

Low feed intake sows gave birth to lighter pigs than sows fed adequate amounts of feed and as a result of lower milk yields these piglets were also lighter at weaning. The slightly higher rates of

liveweight gain of piglets from restrict fed sows in the growing period (7-60 kg) resulted in pigs being the same age at slaughter (100 kg) as those from high feed intake sows. The apparent catch-up growth of piglets following nutritional restriction has been reported previously (Vermedahl et al., 1969; Lodge and Elliot, 1979). The question of the existence of compensatory gain is a controversial issue and it is not clear from this data whether an increase in feed intake or, improvement in feed conversion efficiency accompany the slight increase in growth rate.

The incidence of osteochondrotic lesions in the elbow joint was similar for pigs from both treatment groups. Average lesion scores for sites in the elbow and distal ulna differed inconsistently; the scores of the proximal ulna and lateral humeral condyle were higher in pigs from high feed intake sows ($P < 0.05$), while the distal ulna score was greatest in pigs from restricted fed sows ($P < 0.01$). This would indicate a possible difference in the etiology of articular and physeal types of lesions. Lesions of the articular-epiphyseal cartilage have been demonstrated as morphologically distinct from those found in the distal ulna physis (Hill et al., 1985). In addition, the morphology of articular-epiphyseal and physeal cartilage have been shown to differ from each other representing a possible difference in the process of endochondral ossification in each (Carlson et al., 1985). The articular-epiphyseal lesions are first visible in the cartilage of young pigs at a later age than those in the distal ulna physis which have been observed at birth. The lesion areas in the articular-epiphyseal complex involve chondrolysis and are presumed to progress further into degenerative joint disease (Hill et al., 1984a; 1984b). In

physeal lesions viable chondrocytes appear to be producing abnormal matrix proteoglycans (Farnum et al., 1984, 1986). The morphological distinction between physeal and articular-epiphyseal lesions may support an etiological difference or the articular- epiphyseal lesions may simply be a more advanced stage of the lesions found in the physis. It is not clear which of these hypotheses is true.

The increased severity of lesions in the distal ulna of pigs from restricted fed sows and the correlation of the distal ulna lesion score with maternal weight loss over the 4 week lactation ($P > 0.001$) suggests that sow nutrition is important in the development of distal ulna physeal lesions. The effect was not a result of an indirect response to sow nutrition of increased growth rate since growth rate did not correlate with any of the lesion scores from the elbow and distal ulna. It is proposed that the nutritional restriction imposed on the sow during the critical periods of cartilage development in their offspring may interfere with chondrocyte maturation and the production of a healthy matrix, in the distal ulna physis. Olsson (1978; Olsson and Reiland, 1978) suggested that osteochondrosis results from abnormal growth and maturation of chondrocytes. Hill et al. (1985) suggested that the chondrocytes may be inately defective because of the development of lesions at birth. The intracellular accumulation of lipids in cells surrounding the lesion areas is thought to be an indication of energy depletion (Carlson et al., 1986; Kincaid and Lidvall, 1983), and may represent a problem in cartilage nutrition of young maturing pigs. The cartilage of young pigs is dependent on nutrition from the blood and synovial fluid.

The high incidence of lesions in the distal ulna and elbow joints

of pigs from both nutritional treatments suggests that the etiology of both types of lesions is a complex problem. The distribution of lesions between joint sites in the young piglet is random (Hill et al., 1985) but at 6 months of age they appear most severe at only three specific sites. This implies that although the condition may be congenital, factors influencing endochondral ossification from birth to 6 months of age cause accelerated development of osteochondrosis in the knee and elbow.

One of the most frequently implicated causes of osteochondrosis is the stress of rapid growth rate under confinement rearing conditions, (Reiland 1978; Hanssen and Grondalen, 1979). It has been suggested that rapid gain in weight may increase the mechanical stress on cartilage and bone and ultimately result in damage (Grondalen and Grondalen, 1974; Brennan et al., 1987). However, the data from this experiment are in agreement with other studies involving a normal range of growth rates (from 0.30 to 1.00 kg day⁻¹) which also indicate no significant relationship between growth rate and osteochondrosis or locomotory ability (Nakano et al., 1984; Perrin et al., 1978; Fell et al., 1970). In contrast Hanssen and Grondalen (1979) found a direct relationship between growth rate and locomotory ability. However the incidence of severe lameness in this experiment was relatively low and only more advanced osteochondrotic lesions appeared to result in clinical lameness, in agreement with Grondalen (1974d).

The prevalence of lesions on the weight bearing region of the medial humeral condyle and visible signs of wear on the cranio-medial edge of the proximal radius observed in this experiment have been implicated as evidence of the involvement of mechanical wear at least

as a secondary factor in the development of osteochondrosis (Grondalen, 1974e; Brennan et al., 1987).

No sex or treatment differences were observed in the incidence of osteochondrosis or in the uronic acid content of medial humeral condylar cartilage. More advanced lesions of the medial humeral condyle were associated with reduced proteoglycan content demonstrated by lower uronic acid content. This is in agreement with Nakano et al. (1979a) who observed a significant reduction in uronic acid content of severely osteochondrotic cartilage compared to normal in the condyles of the femur (2.65 vs 5.25 % of dryweight). In contrast Simunek and Muir (1972a, 1972b) found no difference in uronic acid content between normal and osteochondrotic cartilage, despite differences in extractability and size of proteoglycan molecules. The reason for this discrepancy is probably due to differences in size of sampling area.

The observed loss of uronic acid from osteochondrotic cartilage represents a large reduction in proteoglycan content and hence load bearing capacity of the cartilage. It is plausible that any additional weight stress would exacerbate the development of these lesions.

In conclusion, severe maternal nutritional restriction over 3 consecutive lactations had no significant effect on the overall incidence of osteochondrosis in the elbow joint of offspring at 100 kg liveweight. The severity of distal ulna physeal lesions was increased in pigs from restricted fed sows suggesting a possible involvement of sow nutrition in physeal lesions and perhaps a difference in etiology of physeal and articular-epiphyseal lesions.

Table II.1. The effect of sow nutrition on birth weight and subsequent growth rate of offspring to 100 kg liveweight.

	<u>Sow feed intake</u>		SE	<u>Significance</u>
	High	Low		
Number of pigs	39	41		
Birth weight, kg	1.5	1.0	0.04	**
Weaning weight, kg	7.9	5.9	0.22	**
Finishing weight, kg	79.3	80.2	0.72	*
Slaughter age, days	169	172	0.85	*
Growth Rate, kg day ⁻¹				
Grower(7-60 kg)	0.74	0.77	0.06	*
Finisher(60-100 kg)	0.79	0.81	0.19	NS
<u>Overall period(7-100 kg)</u>	<u>0.77</u>	<u>0.79</u>	<u>0.05</u>	<u>NS</u>

NS no significant difference, $P > 0.05$; * significant at $P < 0.05$;

** significant at $P < 0.01$.

Table II.2. Average lesion scores† for sites in the elbow and distal ulna physis of pigs examined.

Site scored	Sow feed intake		Significance
	High (SE)	Low (SE)	
Medial humeral condyle	1.65 (0.16)	1.66 (0.18)	NS
Lateral humeral condyle	1.55 (0.10)	1.27 (0.07)	*
Proximal radius	1.31 (0.09)	1.21 (0.07)	NS
Proximal ulna	1.31 (0.10)	1.07 (0.03)	*
Distal ulna physis	1.47 (0.09)	2.13 (0.12)	**
Locomotority ability	1.31 (0.11)	1.52 (0.11)	NS

†Scores 1-normal, 2-3-minor to moderate lesions, 4-5-severe lesions.

Values in parentheses are standard errors of means.

NS no significant differences, $P > 0.05$; * significant $P < 0.05$;

** significant at $P < 0.01$.

Table II.3. Uronic acid content (% of dry weight) in cartilage from the medial humeral condyle of pigs from high and low feed intake sows.

Sex	Sow feed intake		Significance
	High	Low	
Male	5.11 (36)	5.12 (42)	NS
Female	5.15 (42)	5.36 (40)	NS

Numbers in parentheses represent number of condyles analysed.

NS no significant ($P>0.05$) difference.

Table II.4. Changes in uronic acid content (% of dry weight) of articular cartilage with increasing lesion score in elbow joints.

Lesion score	Mean uronic acid (% dryweight)	SE
1	5.23 (111)	0.14
2	5.43 (21)	0.26
3	5.33 (13)	0.24
4	4.53 (6)	0.57
5	4.24 (7)	0.48
Average	5.19	0.11

Numbers in parentheses represent number of condyles sampled in score category to calculate mean.

E. BIBLIOGRAPHY

- Bhatnager, R., Christian, R.G., Nakano, T., Aherne, F.X., and Thompson, J.R. 1981. Age related changes in osteochondrosis in swine articular and epiphyseal cartilage: light and electron microscopy. *Can. J. Comp. Med.* 45: 188-195.
- Bitter, T., and Muir, H.M. 1962. A modified uronic acid carbazole reaction. *Anal. Bioch.* 4: 330-334.
- Brandt, K., and Muir, H. 1969. Characterisation of protein-polysaccharides of articular cartilage from mature and immature pigs. *Bioch. J.* 114: 871-876.
- Brennan, J.J., Aherne, F.X., and Thompson, J.R. 1986. Effect of dietary calcium and phosphorus levels on performance, bone bending moment and the severity of osteochondrosis and lameness in boars and gilts slaughtered at 100 or 130 kg. *Can. J. Anim. Sci.* 66:777-790.
- Brennan, J.J., and Aherne, F.X. 1987. Effect of floor type on the severity of foot lesions and osteochondrosis in swine. *Can. J. Anim. Sci.* 67:517-523.
- Brennan, J.J., Aherne, F.X., Thompson, J.R., and Nakano, T. 1987. Effect of increased weight stress on joint integrity, synovial fluid composition and Cathepsin B-like activity of articular cartilage in growing swine. *Can. J. Anim. Sci.* 67:525-533.
- Carlson, C.S., Hilley, H.D., and Henrickson, C.K. 1985. Ultrastructure of normal epiphyseal cartilage of the articular epiphyseal cartilage complex in growing swine. *Am. J. Vet. Res.* 46: 306-313.
- Carlson, C.S., Hilley, H.D., Henrickson, C.K., and Meuten, D.J. 1986. The ultrastructure of osteochondrosis of the articular-epiphyseal complex in growing swine. *Calcif. Tissue. Int.* 38: 44-51.

- Drury, R.A.B., and Wallington, E.A. 1967. Carelton's histological techniques. New York: Oxford University Press.
- Empel, W., Walach-Janiak, M., and Fandrejowski, H. 1980. Influence of intensity of feeding and protein body content on the incidence of osteochondrosis in 6 month old pigs. Ann. Wars. Agric. Univ. Vet. Med. 10: 39-43.
- Farnum, C.E., Wilsman, N.T., and Hilley, H.D. 1984. An ultrastructural analysis of osteochondrotic growth cartilage in growing swine. Vet. Pathol. 21: 141-145.
- Farnum, C.E., and Wilsman, N.J. 1986. Ultrastructural histochemical evaluation of growth plate cartilage matrix from healthy and osteochondrotic swine. Am. J. Vet. Res. 47: 1105-1115.
- Fell, B.F., Leigh, L.C., Wenham, G. and Elsley, F.W.H. 1970. Observations on the incidence of spontaneous lesions of bone in pigs. Br. Vet. J. 126: 306-315.
- Fredeen, H.T., and Sather, A.P. 1978. Joint damage in pigs reared under confinement. Can. J. Anim. Sci. 58: 759-773.
- Ghadiaily, J.A., Ghadiaily, R., and Ghadiaily, F.N. 1977. Long-term results of deep defects in articular cartilage. A scanning electron microscope study. Virchows. Arch. B. Cell. Pathol. 25(2):125-136.
- Grondalen, T. 1974a. Osteochondrosis and arthrosis in pigs. I. Incidence in animals up to 120 kg liveweight. Acta. Vet. Scand. 15: 1-25.
- Grondalen, T. 1974b. Osteochondrosis and arthrosis in pigs. II. Incidence in breeding animals. Acta. Vet. Scand. 15: 26-42.
- Grondalen, T. 1974c. Osteochondrosis and arthrosis in pigs. VI. Relationship to feed level and calcium, phosphorus and protein

- levels in the ration. *Acta. Vet. Scand.* 15: 147-169.
- Grondalen, T. 1974d. Leg weakness in pigs. I. Incidence and relationship to skeletal lesions, feed level, protein and mineral supply, exercise and exterior conformation. *Acta. Vet. Scand.* 15(4): 555-573.
- Grondalen, T. 1974e. Osteochondrosis and arthrosis in pigs. VII. Relationship to joint shape and exterior conformation. *Acta. Vet. Scand.* 15 (Suppl 146): 1-32.
- Grondalen, T., and Grondalen, G. 1974. Osteochondrosis and arthrosis in pigs IV. Effect of overloading on the distal epiphyseal plate of the ulna. *Acta. Vet. Scand.* 15: 53-60.
- Hanssen, J.T., and Grondalen, T. 1979. The effect of different dietary factors on growth and development in young boars and gilts. *Z. Tierphysiol. Tierernahrg u Fultermittelkde.* 42: 65-83.
- Hill, M.A., Ruth, G.R., Hilley, H.D., and Hansgen, D.C. 1984a. Dyschondroplasias, including osteochondrosis in boars between 25 and 169 days of age; histologic changes. *Am. J. Vet. Res.* 45: 903-916.
- Hill, M.A., Hilley, H.D., Freeney, D.A., Ruth, G.R., and Hansgen, D.C. 1984b. Dyschondroplasias, including osteochondrosis in boars between 25 and 169 days of age; radiologic changes. *Am. J. Vet. Res.* 45: 917-925.
- Hill, M.A., Ruth, G.R., Hilley, H.D., Torrison, J.L., Bagent, J.K., and Leman, A.D. 1985. Dyschondroplasias of growth cartilages (osteochondrosis) in crossbred commercial pigs at one and 15 days of age: Radiological, angiomicrographical and hisological findings. *Vet. Rec.* 116: 40-47.

- Kincaid, S.A., and Lidvall, E.R. 1983. Observations on the postnatal morphogenesis of the porcine humeral condyle and the pathogenesis of osteochondrosis. *Am. J. Vet. Res.* 44: 2095-2103.
- Lodge, G.I., and Elliot, J.I. 1979. Influence of bodyweight on subsequent growth of hysterecomy derived pigs. *Can. J. Anim. Sci.* 59: 215-216.
- Lythgoe, E.S. 1987. Metabolic and endocrinological consequences of nutritional stress imposed over successive lactations in primiparous and multiparous sows. University of Alberta. Thesis.
- McDevitt, C.A., and Muir, H. 1976. Biochemical changes in the cartilage of the knee in experimental and natural osteoarthritis in the dog. *J. Bone. Joint. Surg.* 58B: 94-101.
- Nakano, T., Aherne, F.X., and Thompson, J.R. 1979a. Changes in swine knee articular cartilage during growth. *Can. J. Anim. Sci.* 59: 167-169.
- Nakano, T., Aherne, F.X., and Thompson, J.R. 1979b. Effects of feed restriction, sex and diethylstilbestrol on the occurrence of joint lesions with some histological and biochemical studies of the articular cartilage of growing-finishing swine. *Can. J. Anim. Sci.* 59: 491-502.
- Nakano, T., Aherne, F.X., and Thompson, J.R. 1981a. Leg weakness and osteochondrosis in pigs. *Pig News and Information.* 2: 29-34.
- Nakano, T., Aherne, F.X., and Thompson, J.R. 1981b. Effect of housing system on the recovery of boars from leg weakness. *Can. J. Anim. Sci.* 61: 335-342.
- Nakano, T., Thompson, J.R., Aherne, F.X., and Christian, R.G. 1982. Observations of abnormalities and age related changes in the

- anconeal process of swine. Am. J. Vet. Res. 43: 1840-1844.
- Nakano, T., Aherne, F.X., and Thompson, J.R. 1983a. Effect of dietary supplementation of vitamin C on pig performance and the incidence of osteochondrosis in elbow and stifle joints in young growing swine. Can. J. Anim. Sci. 63: 421-428.
- Nakano, T., Thompson, J.R., and Aherne, F.X. 1983b. Growth dependent changes in the content of the collagen crosslink, pyridinoline, in joint cartilage from pigs. Can. J. Anim. Sci. 63: 677-681.
- Nakano, T., Aherne, F.X., Brennan, J.J., and Thompson, J.R. 1984. Effect of growth rate on the incidence of osteochondrosis in growing swine. Can. J. Anim. Sci. 64: 134-146.
- National Academy of Sciences-National Research Council (NAS-NRC) 1979. Nutrient requirements of domestic animals. No 2. Nutrient requirements of swine. Eighth revised edition. NAS-NRC, Washington, DC.
- Olsson, S.E. 1978. Osteochondrosis in domestic animals. Acta. Radiol. (Suppl) 358: 9-14.
- Olsson, S.E., and Reiland, S. 1978. The nature of osteochondrosis in animals. Acta. Radiol. (Suppl) 358: 299-306.
- Perrin, W.R., and Bowland, J.P. 1977. Effects of enforced exercise on the incidence of leg weakness in growing boars. Can. J. Anim. Sci. 57: 245-253.
- Perrin, W.R., Aherne, F.X., Bowland, J.P., and Hardin, R.T. 1978. Effects of age, breed and floor type on the incidence of articular cartilage lesions in pigs. Can. J. Anim. Sci. 58: 129-138.
- Reiland, S. 1978. Pathology of so called leg weakness in the pig. Acta. Radiol. (Suppl) 358: 23-44.

- Rosenberg, L. 1971. Chemical basis for the histological use of Safranin-O in the study of articular cartilage. *J. Bone. Jt. Surgery*. 53(A): 69-82.
- Siffert, R.S. 1981. Classification of the osteochondroses. *Clin. Orthop*. 158: 10-18.
- Simunek, Z., and Muir, H. 1972a. Changes in the protein-polysaccharides of pig articular cartilage during prenatal life, development and old age. *Biochem. J.* 126: 515-523.
- Simunek, Z., and Muir, H. 1972b. Proteoglycans of the knee joint cartilage of young normal and lame pigs. *Biochem. J.* 130: 181-187.
- Steele, R.G.D., and Torrie, J.H. 1980. Principles and procedures of statistics. A biometrical approach, 2nd ed. Mc Graw-Hill Book Co., NY.
- Vermedahl, L.D., Meade, R.J., Hanke, H.E., and Rust, J.W. 1969. Effects of energy intake of the dam on reproductive performance, development of offspring and carcass characteristics. *J. Anim. Sci.* 82: 465-472.
- Walker, T., Fell, B.F., Jones, A.S., Boyne, R., and Elliot, M. 1966. Observations on "leg weakness" in pigs. *Vet. Rec.* 79: 472-479.

III. THE EFFECT OF MIXING STRESS ON THE INCIDENCE OF OSTEOCHONDROSIS IN PIGS; SCANNING ELECTRON MICROSCOPY

A. INTRODUCTION

Osteochondrosis is a non-infectious degenerative joint disease found predominantly in the articular-epiphyseal and metaphyseal growth cartilages of growing animals. It is manifested as a disturbance of endochondral ossification in the cartilage. Osteochondrosis is especially prevalent in the knee and elbow joints of commercial pigs and of particular importance as a possible cause of leg weakness in commercial breeding stock, (Olsson and Reiland, 1978). Much attention has been given to understanding the etiology of osteochondrosis in pigs with the main emphasis being on commercial confinement-rearing conditions and nutrition, but the precise factors in the cause and development of the disease have not been fully elucidated.

The prevalence of osteochondrotic lesions in growing-finishing pigs on the weight bearing regions of the condyles of the knee and elbow joints, and visible signs of wear on the articulating surfaces of the cartilage has prompted a number of authors to implicate mechanical wear as a factor in the development of osteochondrosis, osteoarthrosis and osteochondrosis dissecans (Nakano et al., 1979a, b, 1981; Paatsama et al., 1975; Grondalen 1974a, b; Grondalen and Grondalen, 1974).

Chondrocytes in the articular cartilage of a number of species, including sheep and dogs have been shown to change biosynthetic activity in response to loading and mechanical pressure (Caterson and

Lowther, 1978; Palmoski et al., 1979, 1980; Palmoski and Brandt, 1984). Joint cartilage degeneration has been demonstrated under conditions of both severe overloading in conjunction with over use in sheep (Dekel and Weisman, 1978) and reduced weight bearing in dogs (Palmoski et al., 1980). In pigs, degenerative changes indicative of osteochondrosis have been induced in the cartilage of the distal ulna and articular cartilage by means of immobilisation of one foreleg and loading of pigs with lead saddles (Walker et al., 1966; Grondalen and Grondalen, 1974; Brennan et al., 1987). Hence it has been suggested that the cartilage of rapidly growing pigs is at the upper limit of its weight bearing capacity and that any excess loading may lead to degenerative changes. Under commercial conditions the trauma of mixing and transportation of pigs has been suggested as sufficient mechanical stress to result in traumatic damage to cartilage (Grondalen, 1974b, 1977; Grondalen and Grondalen, 1974; Nakano et al., 1981).

Light and transmission electron microscopy have previously been employed to examine the ultrastructural changes in osteochondrotic cartilage (Farnum et al., 1984; Carlson et al., 1986; Bhatnager et al., 1981), however the surface morphology of degenerative articular cartilage has not previously been examined in pigs. The scanning electron microscope has been successfully utilised for examination of the structure of normal and degenerated articular cartilage of a number of species (humans, cats, rats and sheep) because of its excellent resolution and large depth of field (Redler and Zimmy, 1970; Walton, 1977; Dekel and Weissman, 1978).

The objectives of this experiment were to use scanning electron

microscopy to describe the surface morphology of porcine articular cartilage and, in conjunction with conventional analyses to examine the effects of handling and mixing stress on the development of osteochondrotic lesions in growing-finishing pigs.

B. MATERIALS AND METHODS

Sixteen litters of crossbred (Yorkshire X Landrace) pigs equalised to eight pigs per litter were randomly assigned by litter to one of two pre-weaning treatments and one of four post-weaning treatments from birth, (Table III.1).

Pre-weaning. Pre-weaning treatments involved normal handling or handling with exceptional care. Pigs in group 1 were handled with extreme care during all pre-weaning management procedures (teeth and tail clipping, iron injections and tattooing). Pigs in all other groups were handled normally, without any special care, whilst being caught, processed and returned to their pens. Pigs were weaned at four weeks of age and assigned to post-weaning treatment groups involving various amounts of mixing stress. Pigs which died prior to weaning were replaced with littermates. Two pigs died after weaning and could not be replaced.

Housing. After weaning the pigs were housed in 1.2 m X 1.2 m pens until four months of age. Three different floorings were used throughout this part of the experiment, (punched metal, coated weldmesh, and gravelscreen) and pigs from each treatment group were distributed across all floor types evenly. At four months of age pigs

were moved to 1.2 m X 2.4 m pens with concrete floors and straw bedding.

Diet. Pigs were not allowed access to creep feed during the lactation period. All pigs received standard starter (4-8 weeks), grower (8-16 weeks) and finisher (16-24 weeks) diets containing 19, 16 and 14% crude protein, respectively. Feed and water were freely available throughout the trial. Feed intakes were recorded weekly, on a pen basis and individual pig weights were recorded monthly.

Post-weaning mixing. Pigs in groups 1 and 2 remained unmixed as littermates throughout the duration of the trial. Group 3 and 4 pigs were first mixed at weaning; two pigs from each of groups 3 and 4 were placed together into pens of four (8 pairs of 2 pens). Group 3 pigs were mixed once again at 4 months by swapping pigs equally between pairs of pens within group 3. Group 4 pigs were similarly mixed between pairs of pens at 2, 3, 4 and 5 months of age.

Behavior after mixing was observed for all groups of pigs by the author and activity was scored on a scale of 1 to 4 (1-lying down, 4-fighting). The observer was always visible to the animals and recordings of activity were taken at 5 minute intervals to estimate the proportion of time spent fighting after mixing.

One pig from each pen of pigs in treatments 1, 2, 3 and 4 was randomly selected for slaughter at each of 3, 4, 5 and 6 months of age, resulting in eight pigs per treatment x age group. Immediately after slaughter the elbow and knee joints were removed intact from the left side of each carcass. The joints were opened and residues of synovial

fluid and blood were washed from the articular cartilage using deionised water. From each group of eight pigs per treatment x age group half were used for light microscopy and half for scanning electron microscopy.

Light Microscopy. A transverse section of approximately 2 mm x 10 mm cartilage with subchondral bone was taken from the central weight bearing region of the medial humeral and femoral condyles. Samples were fixed in 10% phosphate buffered formalin (pH 7.0) and were decalcified over a period of 1 week. Following dehydration in ascending concentrations of ethanol specimens were cleared and fixed in paraffin (Drury and Wallington, 1967). Transverse sections 7 μ m thick, were taken and stained with safranin-O, fast green and iron haematoxylin (Rosenberg, 1971).

Scanning electron microscopy (SEM). After removal of synovial fluid and debris from the articular surface transverse sections of cartilage with subchondral bone attached (to reduce shrinkage during preparation) were cut from the central weight bearing region of the medial humeral and femoral condyles. Sections were fixed for 3 days in 3% glutaraldehyde buffered with sodium cacodylate (pH 7.2). Dehydration was carried out using ascending concentrations of ethanol and drying was done by CO₂ replacement in a critical point dryer. The sections were attached by Silver Dag (a suspension of metallic silver in acetone based medium) onto aluminium stubs (1 cm diameter) and coated with a layer of gold (15-20 nm) in an Edwards Sputter coater S150B. The articular surface of specimens was examined under a Philips 505

scanning electron microscope, (gun voltage 15kV), at magnifications between 100 and 4000 times.

Uronic acid. A 1 cm² area of cartilage taken down to the subchondral bone, was removed from the central weight bearing region of all medial humeral and femoral condyles for the determination of uronic acid content. Cartilage segments were dried in acetone, diced and dried at 60° C for 30 min. The samples were then subjected to enzyme digestion using papain (5 mg of twice crystallised enzyme per gram of dry cartilage) for 48 hours, at 60° C (McDevitt and Muir, 1976). The digest was centrifuged and aliquots (100µL) were analysed for uronic acid content by a modified carbazole reaction (Bitter and Muir, 1962).

Cartilage and subchondral bone of the elbow (distal humerus, proximal radius and ulna), distal ulna and knee (distal femur, proximal tibia and patella) joints of each pig were examined for soundness. Evidence of osteochondrosis was assessed on a subjective score of 1 to 5 (1-normal and free of lesions; 5-severe osteochondrotic lesions), (Nakano et al., 1983).

Data were analysed by analysis of variance and significant differences between means assessed using SNK and student's t-test, (Steele and Torrie, 1980). Regression analysis was done to compare soundness scores within the same joint.

C. RESULTS

All pigs remained sound with respect to locomotory ability throughout the experiment. One pig from the mixed group had a rectal

prolapse at 3 months of age, possibly induced by the stress of fighting in a confined space. Pre-weaning handling had no significant effect on average daily gain to weaning at four weeks of age. Handling pre-weaning did not significantly affect the occurrence of osteochondrosis in pigs at 3 months of age.

Average daily gain and daily feed intake (on a pen basis) were not significantly affected by post-weaning mixing stress at any time during the growing and finishing periods, (Table III.2). Pigs from all treatment groups spent a large proportion of their time lying down (80 to 90%) and appeared to become less active with increasing age. Fighting as a result of mixing in groups 3 and 4 was generally more severe when mixing occurred at an older age. Pigs in group 3 and 4 spent an average of 13% and 15% more time fighting than pigs in groups 1 and 2 which did not fight at all, though variation with age at mixing and between pens was quite large. The most severe fighting bouts occurred at 4 months of age for group 3 pigs and at 3 and 4 months of age for group 4 pigs. In general the social order of each pen was restored within the first day after mixing, and usually within an hour most severe attacks had ceased. Fighting behavior was carried out generally by reciprocal mouth-to-neck attacks, sometimes to the point of exhaustion.

Upon visual examination of the knee and elbow joints, lesions characteristic of osteochondrosis were observed in pigs from 3 months of age with greatest frequency in the cartilage of the distal ulna and medial femoral condyle, (38% and 22% of pigs affected, respectively). From 4 months of age the frequency and severity of lesions was greatest in the medial femoral condyle and distal ulna (47% and 25% respectively

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at 6 months). The incidence of osteochondrotic lesions at the three most frequently affected sites scored did not differ significantly between pigs from the four treatment groups. The average lesion score at these three sites and the average lesion score for all sites scored were not affected ($P>0.05$) by post-weaning mixing treatment, (Table III.3). Post-weaning mixing stress had no effect ($P>0.05$) on the uronic acid content of cartilage taken from the weight bearing region of the medial humeral and femoral condyles, (Table III.4), though the variation within treatment groups was large. The uronic acid content of medial femoral condylar cartilage was generally higher than that of the medial humeral condyles, (6.45 and 5.76% of dry weight respectively).

The four different floor types used equally over each treatment group between weaning and six months of age did not affect the incidence of osteochondrosis.

Histologically, osteochondrotic lesions were visible by light microscopy in the articular cartilage of a number of pigs from both pre- and post- weaning treatment groups at 3 months of age. Figure III.1 shows the normal appearance and cellular arrangement of the respective layers of articular-epiphyseal cartilage under light microscopy for comparison. The lesions were characterised by small oval areas of microfracture in the middle to deep zones of the cartilage. In more severe cases the microfracture was situated below surface disturbances accompanied by reduced proteoglycan staining intensity (Fig. III.2). In the articular cartilage of older pigs osteochondrotic lesions were characterised by failed endochondral ossification and cartilage separation from bone. Superficial damage (Fig. III.3) was often associated with severe lesions in the mid-deep zone. There were

no apparent differences in the frequency and characteristics of cartilage lesions between pigs from the different mixing treatments, in agreement with macroscopic observations.

Under the scanning electron microscope at low magnifications the surface of articular cartilage appeared very smooth. At higher magnifications (1000X) numerous small bumps (frequently within depressions) were visible and appeared to be below a surface layer of a fibrous nature. The bumps were oval in shape and approximately 10-15 μ m by 6-10 μ m in size and frequently occurred in groups of two to four (Fig. III.4). These surface bumps were considered to be superficial chondrocytes which were covered by a fine fibrillar layer approximately 1.0-1.5 μ m thick. In cartilage from 5 month old pigs larger surface undulations were visible with chondrocytes and microundulations apparent at higher magnifications. The surface of the articular cartilage was roughened with a moth-eaten appearance and showed evidence of surface cracks and splitting. Cell clustering was present to a greater extent in cartilage from 5 month old pigs and cluster size was larger (>8 cells) compared to cartilage from 3 and 4 month old pigs. Pigs in treatment group 3 showed a tendency towards greater amounts of cell clustering in the knee and elbow joint cartilage than pigs from other treatment groups (Fig. III.5). Although age differences were seen in this regard, there was no marked maturation change in surface morphology of cartilage in pigs from 3 to 6 months of age.

Surface debris seen on all samples of cartilage was identified as residual synovial fluid deposits (Walker et al., 1969) and a film of precipitated protein formed between the fixative and surface

proteoglycans. At 6 months of age two pigs from treatment group 3 (knee only) and one from group 4 (knee and elbow) showed extensive surface damage not seen in any of the cartilage samples from pigs in groups 1 and 2 (no mixing stress). Large areas of the surface fibrillar layer were missing in the damaged cartilage (Fig. III.6), exposing underlying bundles of collagen fibres running in a direction parallel to the articular surface. The larger, deeper fibre bundles had a crimped appearance and seemed to lie in parallel sheets (Figs. III.7 and 8). The extent of damage was estimated to cover between 30 and 50% of the sample area (Fig. III.6). The morphology of the cartilage surrounding damaged areas was apparently normal.

D. DISCUSSION

Mixing pigs in groups 3 and 4 resulted in conflict, but the social order of the newly mixed group was usually quickly re-established (1 day post mixing). It was evident that a newcomer to the group or even a littermate need only have been separated for a short period of time in order to cause hostility and fighting on re-entry. It has previously been demonstrated that only one week of separation was necessary for a pig to cause hostility on returning to a stable group (McBride et al., 1964).

The performance of pigs during the growing-finishing period was unaffected by frequent mixing despite the increased levels of fighting in groups of mixed pigs. This is in agreement with the observations of Friend et al., (1983) although the performance of mixed pigs may be depressed under restricted feeding conditions (Sherritt et al., 1974), and growth rate may be more variable as a consequence of an unstable

social order (McBride et al., 1964).

Pre-weaning handling and post-weaning mixing stress had no consistent effect ($P>0.05$) on the appearance and development of osteochondrotic lesions in the cartilage of knee and elbow joints from pigs between 3 and 6 months of age. Pigs in all four treatment groups had a high incidence of moderate to severe lesions in one or more of the sites scored, increasing in frequency with age (56% to 70% from 3 to 6 months of age, respectively). Neither the increase in mechanical stress as a result of mixing or the removal of mixing stress by littermate grouping appeared to cause a difference in the incidence or severity of osteochondrosis.

The uronic acid content of cartilage from the knee and elbow joints did not differ significantly between treatment groups; values approximated those previously reported for pigs, (Nakano et al., 1979; Brennan et al., 1987). However the scanning electron microscopic evidence of surface degeneration characteristic of osteoarthritis observed only in mixed pigs (50 and 25% of pigs in groups 3 and 4 respectively) suggests that these surface lesions are caused by mechanical trauma. Similarly, increased cell clustering seen in the superficial layer of cartilage from mixed pigs may be a response to mechanical stress (Nakano et al., 1979; Brennan et al., 1987). The progression of surface damage from the removal of the surface fibrillar layer down to the larger fibres of the deeper superficial layer (Figure III.8) was estimated by comparison with the structural arrangement of fibres through the respective layers of articular cartilage previously described (Clarke, 1971c; Barnett et al., 1963; Redler, 1974).

The disappearance of the surface layer of fibres with osteo-

arthrotic damage has been reported by a number of authors to be the result of the action of mechanical stresses on the articular surface (Dekel and Weissman, 1978; Walker et al., 1969; Redler, 1974; Walton, 1977; Stofft and Graff, 1983). The lamina splendens of light microscopy, also described in transmission electron microscopy (Bhatnager et al., 1981) corresponds to the layer of fibres covering the articular cartilage described here by scanning electron microscopy (Figs. III.1 and 6). This layer was approximately 1.0 to 1.5 μm thick in pigs and has been reported to be 0.2 to 0.3 μm thick in rabbits (Barnett et al., 1963). Chemically this layer is composed of adsorbed proteoglycans covering an acellular layer of collagen fibres, and is thought to represent a lubrication cushion, hence the loss of this layer may predispose the cartilage to further degeneration as a consequence of mechanical stress or frictional wear.

Criticism of scanning electron microscopy as a method for determining the surface characteristics of articular cartilage has stemmed from contradictory results due to different preparatory techniques and the suggestion of artefacts, (Gardner and McGillivray, 1971). The pits and bumps describing superficial chondrocytes on the surface of porcine articular cartilage are consistent with those observed in morphological analyses of feline, human and rat cartilage, (Clarke, 1971a, b; Redler, 1974; Barnett, 1963). The unevenness of the articular surface has been considered by some authors to represent a property necessary for normal lubrication of the joint (Walker et al., 1969; Mow et al., 1974). More recently however, morphologists have indicated that in vivo, the articular surface is smooth (Boyde and Jones, 1983; Stofft and Graff, 1983; Helminen et al., 1985).

Considerable shrinkage of tissues occurs as a consequence of the severe preparatory methods used in SEM (Bloebaum and Wilson, 1980). In the present study an attempt to minimise shrinkage was made by the use of the critical point drying technique and maintaining samples with underlying bone attached. Nevertheless some shrinkage was apparent causing the protrusion of cell nuclei (bumps) within shrunken cell lacunae (pits) as well as surface splitting. Post-fixation (prior to glutaraldehyde fixation) in osmium tetroxide or thiocarbonylhydrazide has recently been proposed as a means to overcome the occurrence of such preparation artefacts (Bloebaum and Wilson, 1980; Gattone et al., 1982). Despite the problems encountered here with shrinkage, the uniform preparatory methods maintained throughout the experiment, in conjunction with the results of previous studies (Walker et al., 1969; Dekel and Weissman, 1978) allow definite changes in surface morphology to be identified with a high degree of confidence.

The appearance of osteochondrotic lesions in the articular-epiphyseal cartilage preceded the degenerative changes observed by scanning electron microscopy on the articular surface not seen until 6 months of age suggesting that osteoarthrotic damage may be secondary to the deeper osteochondrotic changes. Light microscopy often revealed surface disruption in conjunction with osteochondrotic damage in pigs as early as 3 months of age, therefore it is unclear what the association between osteochondrotic and arthrotic changes is. The mechanical stress of frequent mixing did not appear to have a primary role in the onset of osteochondrosis. Since the relationship between the development of osteochondrosis and the development of osteoarthrosis is unclear, a secondary role for mechanical stress in

osteochondrosis can not be ruled out.

The effects of joint loading and movement on the articular cartilage have been studied in a number of other species using excessive weight bearing (Caterston and Lowther, 1978; Tammi et al., 1983), overloading (Dekel and Weissman, 1978), unloading (Palmoski et al., 1980) and physical exercise (Kincaid and Van Sickle, 1982; Jurvelin et al., 1985; Stofft and Graff, 1983; Paukkonen et al., 1985). With varying degrees of loading the response to mechanical stress varies from a favourable adaptation in the cartilage proteoglycan content (Palmoski et al., 1979; 1980; Paatsama et al., 1975) to degenerative changes (Dekel and Weissman, 1978; Vasan, 1983), suggesting that the chondrocytes are responsive to changes in joint loading above and below a threshold level, whereafter degeneration occurs.

The increasing incidence and severity of degenerative joint disease in pigs with advancing age has prompted suggestions that the cartilage of rapidly growing pigs is approaching this upper threshold level (Brennan et al., 1987). In support of this hypothesis osteochondrotic lesions have been induced by joint overloading in the articular cartilage (Brennan et al., 1987) and distal ulna physis (Grondalen and Grondalen, 1974; Walker et al., 1966) of growing pigs. In both these cases unphysiological levels of loading were used and the non-mechanically stressed control animals also showed a high incidence of osteochondrotic lesions. This implies a more complex etiology for the development of osteochondrosis but suggests that mechanical stresses may be important in the progression of lesions into degenerative joint disease with advancing age.

In conclusion, the degree of mixing employed in this experiment suggests that mixing practices commonly employed on commercial farms may contribute to the development of osteoarthrotic damage, but that the relationship to osteochondrosis remains unclear.

Table III.1. Arrangement of pre- and post-weaning treatment groups.

	Treatment group			
	1	2	3	4
<u>Number of pigs</u>	32	30	32	32
<u>Pre-weaning</u>	Gentle	Normal	Normal	Normal
<u>Birth to 4 weeks</u>	Handling	Handling	Handling	Handling
<u>Post-weaning</u>	Not	Not		
<u>1 to 2 months</u>	Mixed	Mixed	Mixed	Mixed
<u>2 to 3 months</u>	From	From		Mixed
<u>3 to 4 months</u>	Weaning	Weaning		Mixed
<u>4 to 5 months</u>	To	To	Mixed	Mixed
<u>5 to 6 months</u>	Slaughter	Slaughter		Mixed

Table III.2. Performance data for grower and finisher periods.

	<u>Treatment group</u>					
	1	2	3	4	SE	Sig
<u>Performance</u>						
Number of pigs	32	30	32	32		
<u>Grower period (8-60 kg)</u>						
Initial wt.kg	1.21	1.33	1.26	1.30	0.06	NS
Final wt.,kg	58.15	59.65	54.57	57.02	2.17	NS
Avg.daily feed intake,kg	1.43	1.41	1.39	1.40	0.05	NS
Avg.daily gain,kg	0.61	0.60	0.59	0.60	0.03	NS
Feed/gain	2.34	2.36	2.35	2.33	0.02	NS
<u>Finisher period (60-100 kg)</u>						
Initial wt.,kg	58.15	59.65	54.57	57.02	2.17	NS
Final wt.,kg	97.44	93.95	97.66	98.00	5.10	NS
Avg.daily feed intake,kg	2.89	2.16	2.77	2.76	0.16	NS
Avg.daily gain,kg	0.77	0.68	0.77	0.74	0.03	NS
Feed/gain	3.75	3.17	3.62	3.75	0.02	NS
<u>Overall period (8-100 kg)</u>						
Avg.daily feed intake,kg	2.01	1.79	1.96	1.97	0.08	NS
Avg.daily gain,kg	0.65	0.62	0.65	0.65	0.02	NS
Feed/gain	3.09	2.88	3.01	3.03	0.01	NS

NS, no significant difference, $P < 0.05$.

Table III.3. Average lesion scores† for osteochondrosis in the medial humeral and femoral condyles and distal ulnae of pigs from 3 to 6 months of age.

Age (Months)	Site	Treatment group				SE	Sig
		1	2	3	4		
3	Medial humeral condyle	1.75	1.50	1.50	1.75	0.22	NS
	Distal ulna physis	2.12	2.50	2.25	2.88	0.32	NS
	Medial femoral condyle	1.88	1.62	2.38	1.75	0.33	NS
4	Medial humeral condyle	1.87	1.50	2.12	1.25	0.37	NS
	Distal ulna physis	1.38	2.00	1.88	2.12	0.39	NS
	Medial femoral condyle	2.12	2.62	2.38	2.00	0.24	NS
5	Medial humeral condyle	1.50	1.12	1.87	1.00	0.26	NS
	Distal ulna physis	1.62	1.75	2.37	1.62	0.26	NS
	Medial femoral condyle	2.38	2.62	2.38	2.50	0.25	NS
6	Medial humeral condyle	1.25	1.00	1.37	1.00	0.15	NS
	Distal ulna physis	1.62	2.00	1.88	2.00	0.36	NS
	Medial femoral condyle	3.75	1.67	2.88	2.25	0.48	*

NS, no significant difference, $P < 0.05$; * significant at $P < 0.05$.

†score 1-normal; 5-severe osteochondrotic lesions.

Table III.4. Uronic acid content† of articular cartilage in knee and elbow.

Treatment	<u>Medial humeral condyle</u>					<u>Medial femoral condyle</u>				
Group	1	2	3	4	SE	1	2	3	4	SE
<u>Age (m)</u>										
3	5.75	5.22	5.81	6.85	0.62	5.78	5.23	5.86	5.75	0.44
4	6.39	7.04	4.96	5.85	0.89	6.72	6.28	6.76	7.37	0.47
5	5.66	5.83	6.19	5.18	0.57	7.18	6.75	6.91	6.44	0.36
6	5.82	4.56	5.55	4.59	0.48	6.05	6.69	6.89	6.58	0.49

†percent of dry weight.

Plate III.1. Light micrograph showing the normal arrangement of the lamina splendens (LS), articular cartilage (A), zones of the epiphyseal cartilage (E), and subchondral bone (B), (X 265).



Plate III.2. Superficial and underlying damage to the articular cartilage showing reduced proteoglycan staining in the mid zone (arrow). Elbow of pig at 5 months of age (X 120).



Plate III.3. Articular surface disruption in the articular cartilage
of the medial humeral condyle of a pig at 4 months of age (X 470).



Plate III.4. Normal appearance of the articular surface of the medial humeral condyle under scanning electron microscopy (5 months of age). Chondrocytes appear in clusters of four (C) (X 970).



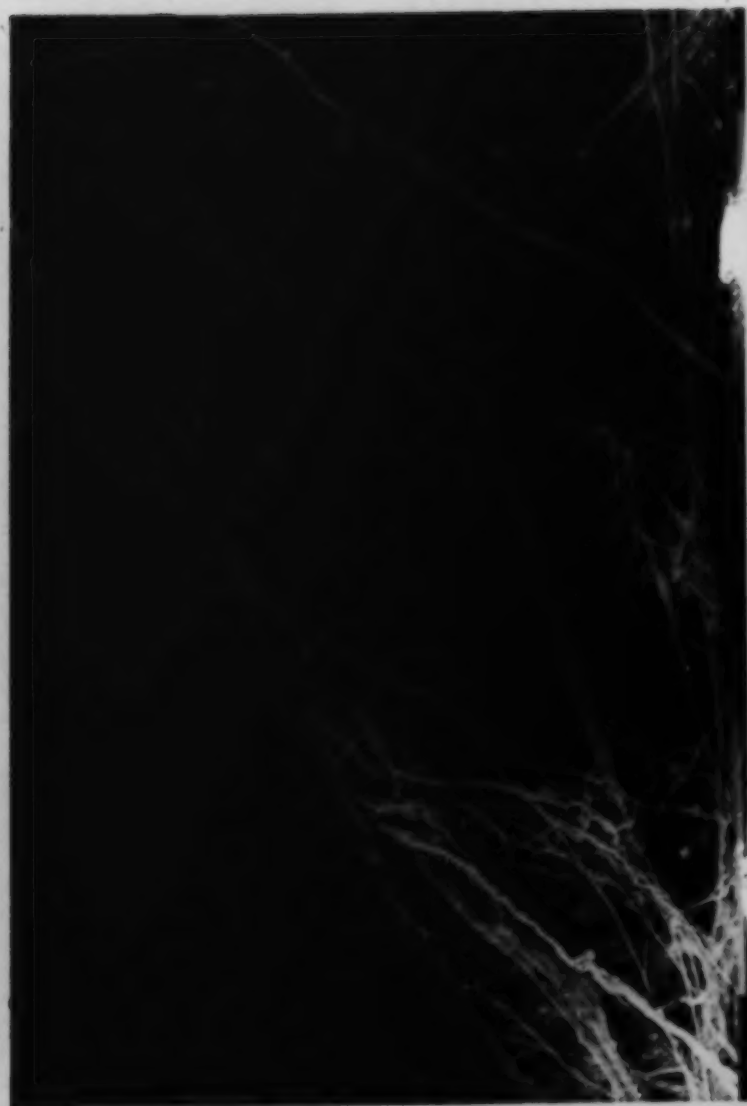
Plate III.5. Articular surface of the medial humeral condyle of a pig
at 5 months of age showing increased surface chondrocyte clustering and
cluster size (CC) (X 920).



Plate III.6. Extensive area of surface disruption on the medial humeral condyle of a pig at 6 months of age, showing disappearance of surface layer (arrow) (X 85).



Plate III.7. Area of extensive surface wear revealing underlying collagen fibre arrangement (F) parallel to the articular surface, and adjacent normal surface appearance (N) (X 425).



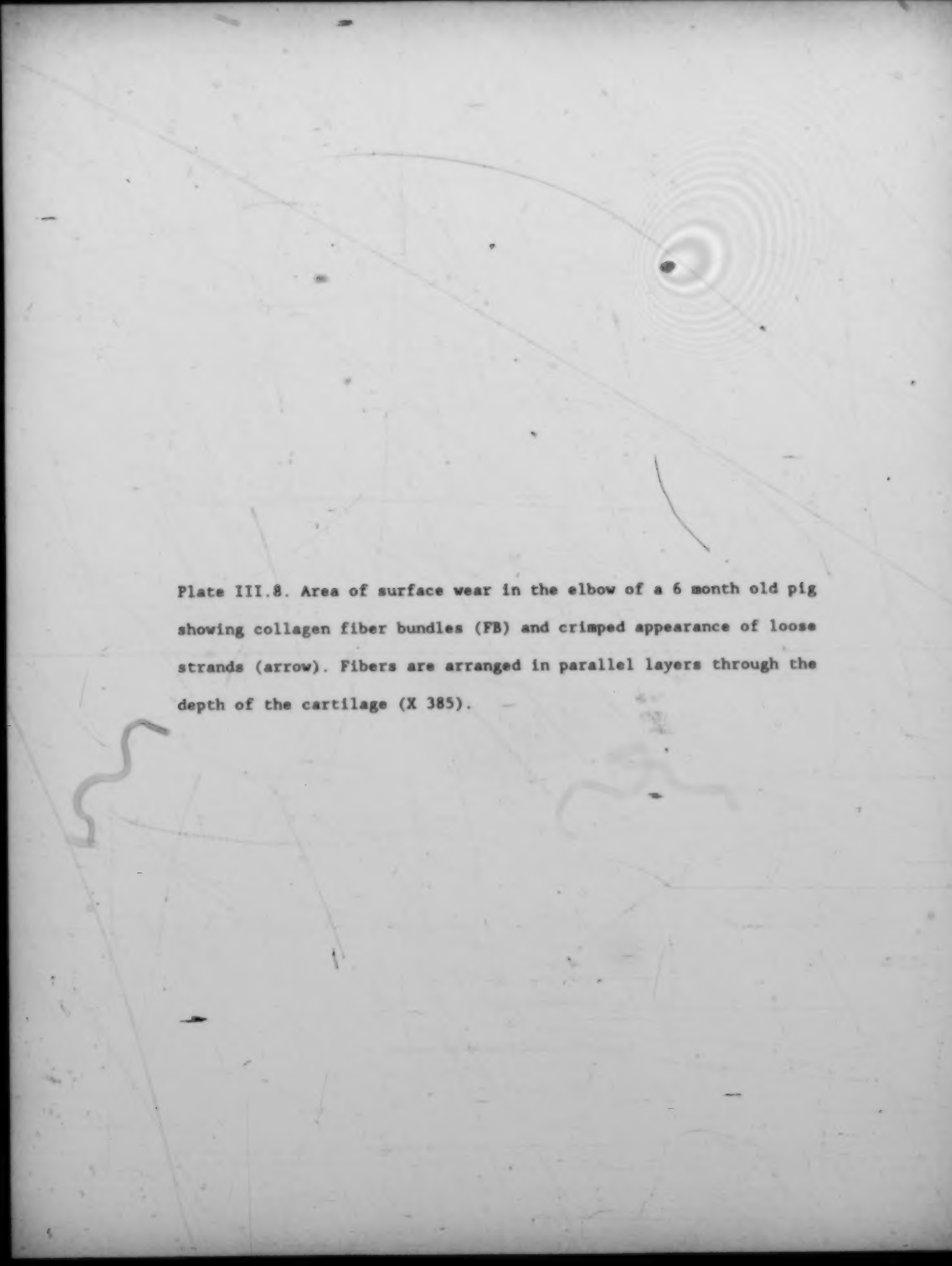
A black and white micrograph showing a dense network of collagen fibers. A prominent feature is a circular, ripple-like structure in the upper right quadrant, possibly representing a focal point of stress or a specific fiber arrangement. A long, thin, slightly curved line runs diagonally across the upper half of the image. In the lower left, there is a distinct, wavy, S-shaped structure. The overall texture is granular and fibrous.

Plate III.8. Area of surface wear in the elbow of a 6 month old pig showing collagen fiber bundles (FB) and crimped appearance of loose strands (arrow). Fibers are arranged in parallel layers through the depth of the cartilage (X 385).



E. BIBLIOGRAPHY.

- Barnett, C.H., Cochrane, W., and Palfrey, A.J. 1963. Age changes in articular cartilage of rabbits. *Annals. Rheum. Dis.* 22: 389-399.
- Bhatnager, R., Christian, R.G., Nakano, T., Aherne, F.X., and Thompson, J.R. 1981. Age related changes in osteochondrosis in swine articular epiphyseal cartilage: light and electron microscopy. *Can. J. Comp. Med.* 45: 188-195.
- Bitter, T., and Muir, H.M. 1962. A modified uronic acid carbazole reaction. *Anal. Biochem.* 4: 330-334.
- Bloebaum, R.D., and Wilson, A.S. 1980. The morphology of the surface of articular cartilage in adult rats. *J. Anat.* 131 (1-2): 333-346.
- Brennan, J.J., Aherne, F.X., Thompson, J.R., and Nakano, T. 1987. Effect of increased weight stress on joint integrity, synovial fluid composition and Cathepsin B-like activity of articular cartilage in growing swine. *Can. J. Anim. Sci.* 67:525-533.
- Carlson, C.S., Hilley, H.D., Henrickson, C.K., and Meuten, D.J. 1986. The ultrastructure of osteochondrosis of the articular- epiphyseal cartilage complex in swine. *Calcif. Tiss. Int.* 38: 44-51.
- Caterson, B., and Lowther, D.A. 1978. Changes in the metabolism of the proteoglycans from sheep articular cartilage in response to mechanical stress. *Biochim. Biophys. Acta.* 540: 412-422.
- Clarke, I.C. 1971a. Surface characteristics of human articular cartilage - a scanning electron microscope study. *J. Anat.* 108: 23-30.
- Clarke, I.C. 1971b. Human articular surface contours and related surface depression frequency studies. *Annals. Rheum. Dis.* 30: 15-23.

- Clarke, I.C. 1971c. A review and scanning electron microscope study. 1. The interterritorial fibrillar architecture. *J. Bone. Joint. Surg.* 52B: 732-750.
- Dekel, S., and Weissman, S.L. 1978. Joint changes after overuse and peak overloading of rabbit knees in vivo. *Acta. Orthop. Scand.* 49: 519-528.
- Drury, R.A.B., and Wallington, E.A. 1967. Cargill's histological techniques. N.Y. Oxford University Press.
- Farnum, C.E., Wilsman, N.T., and Hilley, H.D. 1984. An ultrastructural analysis of osteochondrotic growth cartilage in growing swine. *Vet. Path.* 21: 141-145.
- Friend, T.H., Knabe, D.A., Tanksley, T.D. 1983. Behavior and performance of pigs grouped by three different methods at weaning. *J. Anim. Sci.* 57: 1406-1411.
- Gardner, D.L., and McGillivray, D.C. 1971. Surface structure of articular cartilage. Historical review. *Ann. Rheum. Dis.* 30: 10-14.
- Gattone, V.H., Saul, F.P., O'Conner, B.L., and McNamara, M.C. 1982. Scanning electron microscopic study of age related surface changes in rat femoral head articular cartilage. *J. Submicrosc. Cytol.* 14 (1): 99-106.
- Grondalen, T. 1974a. Osteochondrosis and arthrosis in pigs VII. Relationship to joint shape and exterior conformation. *Acta. Vet. Scand.* 15 (Suppl 46): 1-32.
- Grondalen, T. 1974b. Osteochondrosis, arthrosis and leg weakness in pigs. *Nord. Vet. Med.* 26: 534-537.
- Grondalen, T., and Grondalen, G. 1974. Osteochondrosis and arthrosis in pigs. IV. Effects of overloading on the distal epiphyseal plate of

- the ulna. *Acta. Vet. Scand.* 15: 53-60.
- Grondalen, T. 1977. View points on the porcine leg weakness syndrome. *Proceedings of the 3rd Int. Conf. on Production Diseases in Farm Animals, Wageningen, The Netherlands. 1976.* pp 214-217.
- Helminen, H.J., Jurvelin, J., Tammi, M., Pelttari, A., Svartback, C.M., Kiviranta, I., Saamanen, A.M., and Paukkonen, K. 1985. Prolonged ethanol replacement by CO₂ increases splits on articular cartilage surface after critical point drying. *J. Microsc. (Oxf)* 137(3): 305-312.
- Jurvelin, J., Kuusela, T., Heikkila, R., Pelttari, A., Kiviranta, I., Tammi, M., and Helminen, H.J. 1983. Investigation of articular cartilage surface morphology with a semiquantitative scanning electron microscopy method. *Acta. Anat.* 116: 302-311.
- Kincaid, S.A., and Van Sickle, D.C. 1982. Effects of exercise on the histochemical changes of articular chondrocytes in adult dogs. *Am. J. Vet. Res.* 43: 1218-1226.
- McBride, G., James, J.W., and Hodgins, N. 1964. Social behaviour of domestic animals, IV. Growing pigs. *An. Prod.* 6 (II): 129-139.
- McDevitt, C.A., and Muir, H. 1976. Biochemical changes in the cartilage of the knee in experimental and natural osteoarthritis in the dog. *J. Bone. Joint. Surg.* 58B: 94-101.
- Mow, Van.C., Lai, W.M., Glesenfeld, J., and Redler, J. 1974. Some surface characteristics of articular cartilage. II. On the stability of articular cartilage and a possible biomechanical factor in etiology of chondrodegeneration. *J. Biomechanics.* 7: 457-466.

- Nakano, T., Aherne, F.X., and Thompson, J.R. 1979a. Changes in swine knee articular cartilage during growth. *Can. J. Anim. Sci.* 59: 167-169.
- Nakano, T., Aherne, F.X., and Thompson, J.R. 1979b. Effects of feed restriction, sex and diethylstilbestrol on the occurrence of joint lesions with some histological and biochemical studies of the articular cartilage of growing-finishing swine. *Can. J. Anim. Sci.* 59: 491-502.
- Nakano, T., Aherne, F.X., and Thompson, J.R. 1981. Leg weakness and osteochondrosis in pigs. *Pig. News and Info.* 2:29-34.
- Nakano, T., Thompson, J.R., and Aherne, F.X. 1983. Growth dependent changes in the content of the collagen crosslink pyridinoline in joint cartilage from pigs. *Can. J. Anim. Sci.* 63: 677-681.
- Olsson, S.E., and Reiland, S. 1978. The nature of osteochondrosis in animals. *Acta. Radiol. (Suppl)* 358: 299-306.
- Paatsama, S., Rokkanen, P., and Jussila, J. 1975. Etiological factors in osteochondritis dissecans. *Acta. Orthop. Scan.* 46: 906-918.
- Palmoski, M.J., Perricone, E., and Brandt, K.D. 1979. Development and reversal of a proteoglycan aggregation defect in normal canine knee cartilage after immobilisation. *Arthritis. Rheum.* 22: 508-517.
- Palmoski, M.J., Colyer, R.A., Brandt, K.D. 1980. Joint motion in the absence of normal loading does not maintain normal articular cartilage. *Arthritis. Rheum.* 23: 325-334.
- Palmoski, M.J., and Brandt, R.D. 1984. Effects of static and cyclic compressive loading on articular cartilage plugs in vitro. *Arthritis. Rheum.* 27: 675-681.

- Paukkonen, K., Selkkinen, K., Jurvelin, J., Kiviranta, I., and Helminen, H.J. 1985. Cells and nuclei of articular cartilage chondrocytes in young rabbits enlarged after non-strenuous physical exercise. *J. Anat.* 142: 13-20.
- Redler, I., and Zimmer, M.L. 1970. scanning electron microscopy of normal and abnormal articular cartilage and synovium. *J. Bone. Joint. Surg.* 52A 1395.
- Redler, I. 1974. A scanning electron microscope study of human normal and osteoarthritic articular cartilage. *Clin. Orthop.* 103: 262-268.
- Rosenberg, L. 1971. Chemical basis for the histological use of safranin-O in the study of articular cartilage. *J. Bone. Jt. Surg.* 53A: 69-82.
- Sherritt, G.W., Graves, H.B., Gobble, J.L., and Hazlett, V.E. 1974. Effects of mixing pigs during the growing-finishing period. *J. Anim. Sci.* 39: 84-837.
- Steele, R.G.D., and Torrie, J.H. 1980. Principles and procedures of statistics. A biometrical approach. 2nd ed. McGraw-Hill Book Co., NY.
- Stofft, E., and Graf J. 1983. Rasterelektronenmikroskopische Untersuchung des hyalinen Gelenkknorpels. *Acta. Anat.* 116 (2): 114-125.
- Tammi, M., Saamanen, A-M., Jauhiainen, A., Malmgren, O., Kiviranta, I., and Helminen, H. 1983. Proteoglycan alterations in rabbit knee articular cartilage following physical exercise and immobilisation. *Conn. Tiss. Res.* 11: 45-55.
- Vasan, N. 1983. Effects of physical stress on the synthesis and degradation of cartilage matrix. *Conn. Tiss. Res.* 12: 49-58.

Walker, P.S., Sikorski, J., Dowson, D., Longfield, M.D., Wright, V.,
and Buckley, T. 1969. Behaviour of synovial fluid on surfaces of
articular cartilage. A scanning electron microscope study. Ann.
Rheum. Dis. 28: 1-14.

Walker, T., Fell, B.F., Jones, A.S., Boyne, R., and Elliot, M. 1966.
Observations on "leg weakness" in pigs. Vet. Rec. 79: 472-479.

Walton, M. 1977. Studies of degenerative joint disease in the mouse
knee joint; scanning electron microscopy. J. Pathol. 123 (4): 211.

IV. GENERAL DISCUSSION

Restriction of maternal energy and protein supply over three consecutive lactations caused a decrease in the birth weight and weaning weight of offspring. These pigs had a slightly higher rate of liveweight gain during the growing period relative to pigs from sows fed adequate levels. Maternal nutritional regime did not significantly affect the overall incidence and severity of osteochondrosis in their offspring at 100 kg liveweight.

Nutritional restriction of sows caused an increase in the severity of physeal lesions in the distal ulna of offspring suggesting the involvement of maternal nutrition in the early appearance of physeal lesions in relation to articular-epiphyseal complex cartilage lesions (Hill et al., 1984). The results of this study, in conjunction with previously reported morphological distinctions between cartilage lesions in the articular-epiphyseal complex and physis (Hill et al., 1985; Carlson et al., 1985), suggests a possible difference in the etiology of lesions in the two cartilage sites.

It has been suggested that maternal nutrition may influence the development and maturation of chondrocytes (Olsson, 1978) and the process of endochondral ossification. The high incidence of osteochondrosis in pigs from both sow nutrition groups and the development of severe lesions in specific sites of the knee and elbow joints indicate a more complex etiology for the disease.

The weight stress of rapid rates of liveweight gain achieved by commercial pigs has frequently been proposed as a factor in the

development of osteochondrosis in growing-finishing pigs (Reiland, 1978). In agreement with previous reports (Nakano et al., 1984) the large range of growth rates observed in the experiment reported in chapter II were poorly correlated with the incidence and severity of the disease. The prevalence of lesions on the central weight bearing regions of the medial humeral and femoral condyles in conjunction with visible signs of wear on the cranio-medial edge of the proximal radius were indicative of the involvement of mechanical factors in the development of osteochondrosis. Flattening of the femoral head and correlation of lesion score on the medial humeral condyle with the incidence of wear on the proximal radius have also been suggested to be the result of mechanical stress (Nakano et al., 1981).

The experiment described in chapter III was designed to examine the influence of mixing stress on joint cartilage integrity. Frequent mixing of pigs in the growing-finishing period did not significantly affect the incidence or severity of osteochondrosis. Fighting as a result of mixing caused superficial osteoarthrotic damage but the relationship between this and underlying osteochondrotic damage was unclear. It is possible that either surface degeneration may lead to the loss of proteoglycans from the mid zone and osteochondrotic damage, or underlying osteochondrotic damage may weaken the ability of the superficial layer to resist frictional wear. The order and relationship of osteochondrotic and arthrotic damage is as yet unclear.

Trauma has been postulated as a major factor in the development of osteochondrosis, (particularly osteochondrosis dissecans) (Paatsama et al., 1975) and osteoarthrosis in many species, especially in humans.

(Douglas and Rang, 1981). Cartilage chondrocytes demonstrate changed biosynthesis in response to mechanical pressure (Vasan, 1983) and degeneration has been induced by excessive over use and overload (Dekel and Weissman, 1978) and by reduced joint motion and loading (Palmoski et al., 1980). The mechanism of chondrocyte response to pressure is unknown. It has been suggested that mechanical stress may cause direct cartilage disruption particularly at sites of weakness such as cartilage canals and interruption of metaphyseal blood supply (Kincaid et al., 1985; Walker et al., 1966; Grondalen and Grondalen, 1974). Alternatively, mechanical pressure may cause elevated release of lysosomal enzymes (cathepsins and proteases) (Ali and Evans, 1973) or production of cell messengers (cAMP and prostaglandins) leading to excessive cartilage breakdown (Dekel and Weissman, 1978).

It is evident that mechanical pressure is an important factor in the maintenance of cartilage integrity and subtle differences between physiological and excessive levels of joint motion and loading exist. It will be necessary to further characterise the response of porcine cartilage to physiological and non-physiological loading conditions and also to examine chemical and hormonal changes in disease situations.

Non-invasive methods of determining the presence of osteochondrosis would provide a better means of following the development and progression of the disease and provide information on the possibilities for recovery.

A. BIBLIOGRAPHY

- Ali, S.Y., and Evans, L. 1973. Enzymatic degradation of cartilage in osteoarthritis. *Fed. Proc.* 32: 1494-1498.
- Carlson, C.S., Hilley, H.D., and Henrickson, C.K. 1985. Ultrastructure of normal epiphyseal cartilage of the articular-epiphyseal cartilage complex in growing swine. *Am. J. Vet. Res.* 46: 306-313.
- Dekel, S., and Weissman, S.L. 1978. Joint changes after overuse and peak overloading of rabbit knees in vivo. *Acta. Orthop. Scand.* 49: 519-528.
- Douglas, G., and Rang, M. 1981. The role of trauma in the pathogenesis of the osteochondroses. *Clin. Orthop.* 158: 28-32.
- Grondalen, T., and Grondalen, G. 1974. Osteochondrosis and arthrosis in pigs IV. Effect of overloading on the distal epiphyseal plate of the ulna. *Acta. Vet. Scand.* 15: 53-60.
- Hill, M.A., Ruth, G.R., Hilley, H.D., and Hansgen, D.C. 1984. Dyschondroplasias, including osteochondrosis in boars between 25 and 169 days of age; histologic changes. *Am. J. Vet. Res.* 45: 903-916.
- Hill, M.A., Ruth, G.R., Hilley, H.D., Torrison, J.L., Bagent, J.K., and Leman, A.D. 1985. Dyschondroplasias of growth cartilages (osteochondrosis) in crossbred commercial pigs at one and 15 days of age: Radiological, angiomicrographical and histological findings. *Vet. Rec.* 116: 40-47.
- Kincaid, S.A., Allhands, R.V., and Pijanowski, G.J. 1985. Chondrolysis associated with cartilage canals of the epiphyseal cartilage of the distal humerus of growing pigs. *Am. J. Vet. Res.* 46: 726-732.

- Nakano, T., Aherne, F.X., and Thompson, J.R. 1981. Leg weakness and osteochondrosis in pigs. *Pig. News. Info.* 2: 29-34
- Nakano, T., Aherne, F.X., Brennan, J.J., and Thompson, J.R. 1984. Effect of growth rate on the incidence of osteochondrosis in growing swine. *Can. J. Anim. Sci.* 64: 134-146.
- Olsson, S.E. 1978. osteochondrosis in domestic animals. *Acta. Radiol.* (Suppl) 358: 9-14.
- Paatsama, S., Rokkanen, P., and Jussila, J. 1975. Etiological factors in osteochondritis dissecans. *Acta. Orthop. Scand.* 46: 906-918.
- Palmoski, M.J., Colyer, R.A., and Brandt, K.D. 1980. Joint motion in the absence of normal loading does not maintain normal articular cartilage. *Arthritis. Rheum.* 23: 325-334.
- Reiland, S. 1978. Pathology of so called leg weakness in the pig. *Acta. Radiol.* (Suppl) 358: 23-44.
- Vasan, N. 1983. Effects of physical stress on the synthesis and degradation of cartilage matrix. *Conn. Tissue. Res.* 12: 49-58.
- Walker, T., Fell, B.F., Jones, A.S., Boyne, R., and Elliot, M. 1966. Observations on "leg weakness" in pigs. *Vet. Rec.* 79: 472-479.